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(FILE 'HOME' ENTERED AT 08:45:26 ON 23 NOV 2008)

FILE 'MEDLINE, SCISEARCH, CAPLUS, BIOSIS' ENTERED AT 08:45:45 ON 23 NOV 102631 S VEGF? OR VEGF-165 OR VEGF1? OR VEGF1-165 OR VEGF (2W) 165 L1L2 200757 S CARDIOMYO? L3 761 S L1 (L) L2 L4326 DUP REM L3 (435 DUPLICATES REMOVED) L554 S L4 AND PY<=2002 12 S L5 AND (PLASMID? OR VECTOR? OR VIRUS?) L6 12 SORT L6 PY L7 E LEGUENS RUBEN?/AU E LAGUENS RUBEN?/AU L8 50 S E1 L9 11 S L8 AND L2 L10 4 DUP REM L9 (7 DUPLICATES REMOVED

- L7 ANSWER 7 OF 12 MEDLINE on STN
- TI Entrance in mitosis of adult cardiomyocytes in ischemic pig hearts after **plasmid**-mediated rhVEGF165 gene transfer.
- SO Gene therapy, (2002 Dec) Vol. 9, No. 24, pp. 1676-81. Journal code: 9421525. ISSN: 0969-7128.
- AU Laguens R; Cabeza Meckert P; Vera Janavel G; Del Valle H; Lascano E; Negroni J; Werba P; Cuniberti L; Martinez V; Melo C; Papouchado M; Ojeda R; Criscuolo M; Crottogini A
- Replacement of the cell loss occurring after acute myocardial infarction AB has been proposed as a potential treatment to prevent heart remodeling and failure. On account that cardiomyocytes express VEGF receptors and that **VEGF** triggers mitogen-activated protein kinases, we investigated if ${f VEGF}$ gene transfer may induce cardiomyocyte replication. In a pig model of chronic myocardial ischemia achieved by Ameroid occlusion of the left circumflex coronary artery, we observed that direct intramyocardial injection of a plasmid encoding human VEGF(165) induced a several-fold increase in cardiomyocyte mitotic index and in the number of cardiomyocyte nuclei per unit volume as compared with pigs receiving plasmid devoid of gene. Despite images of conventional cytokinesis were not observed, the fact that caryokinesis is an obligatory step for cell division suggests that our finding may contribute to the issue of heart regeneration and may potentially widen the therapeutic spectrum of **VEGF** gene transfer.